Minutes of the Dioxin Reassessment Review Subcommittee Meeting

November 1-2, 2000

U.S. Environmental Protection Agency
Science Advisory Board
Ramada Plaza Pentagon
Alexandria, VA

The Dioxin Reassessment Review Committee (DDRC) of the US EPA Science Advisory Board (SAB) Executive Committee met on Wednesday and Thursday, November 1-2, 2000, at the Ramada Plaza Hotel Pentagon, 4641 Kenmore Avenue, Alexandria, VA. The meeting was announced in the Federal Register at FR Vol. 65, Number 196, October 10, 2000, pages 60190-60192 (Attachment A). The proceedings followed the agenda (Attachment B) with some deviations to accommodate various individual's scheduling problems.

In April 1991, EPA announced that it would conduct a scientific reassessment of the potential health risks of exposure to dioxin and related compounds. A multi-volume document titled ``Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds" was published in 1994. In 1995, this draft was reviewed by EPA's Science Advisory Board (SAB), which issued a report (EPA-SAB-EC-95-021) stating that there was no need for further SAB review of the exposure sections, but that EPA should develop a new chapter on toxicity equivalence factors (TEFs), revise the sections addressing Dose Response Modeling, and the Risk Characterization, and conduct external peer review of these three sections before returning to the SAB for another review. EPA subsequently revised the document, and conducted external peer reviews as recommended. The Agency then requested that the SAB review the revised reassessment document. The Dioxin Reassessment Review Committee consequently met on November 1 and 2, 2000, in Alexandria, Virginia, to address a Charge comprising 20 primary questions on the above noted issues.

Wednesday, November 1, 2000

Convene the Meeting: Dr. Morton Lippmann convened the meeting at 8:45 a.m. and welcomed all the attendees. He than commented briefly on the history of the dioxin reassessment and the task before the Committee.

The following Members, Consultants, and Federal Expert served on the Committee: Dr. Morton Lippmann (Member and Chair); Drs. George Lambert and Valerie Thomas, (Members); Drs. Roy Albert, Stephen Brown, Richard Clapp, Kenny S. Crump, John Graham, William Greenlee, Nancy Kim, Kai-shen Liu, Gene Matanoski, Ernest McConnell, Thomas McKone, Maria Morandi, Dennis

Paustenbach, Gary Perdew, Knute Ringen, and Bernard Weiss (Consultants); and Drs. Michael Luster and Thomas Umbreit (Federal Experts). Drs. Luster, McConnell, and Ringen were unable to attend the public meeting but were asked to participate in the review of the Committee's report. Mr. Samuel Rondberg served as the Committee Designated Federal Officer. The Committee roster is provided as Attachment C.

After a brief discussion of administrative issues and the Federal Advisory Committee Act (FACA) and its requirements by the Designated Federal Officer (DFO), the Chair asked each Member, Consultant, and Federal Expert on the Subcommittee to identify him/her self, their organizational affiliation, research interests and sources of support, and to state if they had identified any possible conflict of interest concerning the matters to be discussed by the Committee. No such issues were identified.

Agency staff and public attendees are noted on the sign in sheets (Attachment D)

A transcript of the meeting is incorporated as Attachment E. Because of technical problems with the transcription equipment, the proceedings prior to the presentations by EPA staff were not captured.

Background of the Issues: Following the introduction of the participants on the Subcommittee, EPA staff (Dr. William Farland, Director of the EPA National Center for Environmental Assessment) briefed the Subcommittee on the major issues in the Reassessment (handouts for this presentation are incorporated as Attachment F).

Following the EPA briefing and a brief break, the Subcommittee took comments from the initial group of public commentors (the list of commentors is provided in Attachment G). The Subcommittee then broke for lunch.

Specific Issues Discussion: The Subcommittee reconvened at XXPM and turned to the substantive issues for the review. The following brief paragraphs attempt to capture the overall conclusions (or lack thereof) of the Subcommittee's deliberations on each issue, not every nuance raised in the course of (frequently) lengthy discussions.

Question 1 asked: "Did EPA adequately justify its use of body burden as a dose metric for inter-species scaling? Should the document present conclusions based on daily dose?" Dr. Kai-Shen Liu led the discussion.

The Subcommittee was favorably impressed by the correspondence between the animal data and the human data using the body burden measure, thought that further exploration of the data within humans (rather than from animals to humans) was warranted. The Subcommittee also stated that average body burden is apt not to be a very good measure for extrapolating across different patterns of exposure.

Question 2 asked: "Has EPA's choice of the MOE approach to risk assessment adequately considered

that background levels of dioxins have dropped dramatically over the past decade, and are continuing to decline? How might the rationale be improved for EPA's decision not to calculate an RfD or RfC and for the recommended MOE approach for conveying risk information? Is an MOE approach appropriate as compared to the traditional RfD/RfC? Should the document present an RfD/RfC?" Dr. Brown served as led discussant.

The Subcommittee in general supported EPA's position that setting an RfD or RfC substantially below exposures that would be estimated from current ambient dioxin levels would be essentially meaningless for risk management. The MOE approach would therefore be preferred, at least until exposures estimated from ambient levels of dioxin drop well below the RfD/RfC values that EPA believes are appropriate. Some Members of the Subcommittee believed that the MOE approach would be preferable regardless of the levels of ambient exposure because it more properly leaves decisions about the acceptability of a margin of exposure in the hands of risk managers instead of incorporating them through uncertainty factors in setting the RfD/RfC. A few Members of the Subcommittee saw the dioxin reassessment as an opportunity to use dose response information explicitly in a non-cancer risk assessment, producing estimates of risk at various levels of exposure for various endpoints. Also, some Members expressed concern about the practical consequences of the absence of RfD/RfC information for dioxin and the IRIS data base.

Question 3 asked: "The SAB commented that previous dose response modeling was too limited to biochemical endpoints. Are the calculations of a range of ED_{O1} body burden for non-cancer effects in rodents responsive and clearly presented? Please comment on the weight of evidence interpretation of the body burden data, associated with a one percent rate of non-cancer effects that is presented in Chapter 8, Appendix I, and Figure 8-1." Dr. Crump served as the lead discussant.

The Subcommittee suggested that EPA explore the statistical uncertainty in the dose-response curve shape estimates, and specifically the parameter in the Hill Model which is used as a measure of curve shape. They noted (considering statistical uncertainty in general) that the reassessment document provides ED_{01} values and lower confidence limits, and evaluates the uncertainty by comparing the ED_{01} to the statistical lower bound. But these limits are not symmetric about the point estimate, so comparison of the upper limit to the lower limit would be a much more allowable measure of the uncertainty in the ED_{01}. Regarding whether a one percent risk is appropriate for defining the ED, The Subcommittee felt that this is primarily a policy decision. EPA has generally used 10 percent in the past, and the Members saw no over-riding reason to use a different value for different chemicals. The Subcommittee recommended that EPA articulate a consistent policy. It might inform this decision if EPA would calculate both point estimates of EDs and associated confidence limits, using different risk values, for example; one percent, five percent and ten percent, to see how the statistical uncertainty is affected by this choice.

Question 4 asked: "How might the discussion of mode of action of dioxin and related compounds be improved?" Dr. Umbreit served as the lead discussant.

The Subcommittee found that, in general, the EPA's background chapter on mechanisms of action was excellent. It was noted however, that this particular chapter was brief for such an important topic, and might not present a full enough picture of the major actions and complexities involved. The Members also noted that there was only rather limited discussion of Ah receptor binding in other species (information that might aid in interpreting the human data). Some detail on the extrapolation from rodent data to human effects involving the Ah receptor in the Reassessment document would be thus helpful.

Question 5 asked: "Despite the lack of congener-specific data, does the discussion in the Integrated Summary and Risk Characterization support EPA's inference that these effects may occur for all dioxin-like compounds, based on the concept of toxicity equivalence?" Dr. Albert served as the lead discussant.

The Subcommittee concluded that EPA provided a good discussion and defense of the use of TEQs and TEFs, but noted they have to be applied with caution as they're only an approximation and convenience for handling complex mixtures. As such their application is often best when there is specific context for application, e.g., in a screening scenarios. Using TEQ/TEFs for evaluating exposure levels in epidemiology may be convenient, but it may include the possibility of significant error. The Subcommittee agreed that EPA is within the bounds of current scientific thought and usage when using TEFs in a judicious manner, and should do so until such time as a better approach is developed.

Question 6 asked: "Is the history, rationale, and support for the TEQ concept, including its limitations and caveats, laid out by EPA in a clear and balanced way in Chapter 9? Did EPA clearly describe its rationale for recommending adoption of the 1998 WHO TEFs." Dr. Weiss served as the lead discussant.

The Subcommittee concluded that (even after acknowledging the uncertainties in the concept), given the amount of data and expert opinion leading to the TEF values recommended by the WHO, it makes the most scientific sense for EPA to adopt the same TEQ values. One caveat raised by a Member -- EPA needs to consider whether effective TEQs are the same through the human developmental cycle, and that a fetus or a child may not have the same TEQ as an adult.

Question 7 asked: "Does EPA establish clear procedures for using, calculating, and interpreting toxicity equivalence factors? Dr. Paustenbach served as the lead discussant.

The Subcommittee agreed that the EPA did an excellent job of summarizing the published work in this area. Based on the quality and number of previous scientific bodies that have evaluated this approach over the years, the Members concluded that the Agency had done a very good job addressing the various concerns about the development and application of the TEF/TEQ procedure expressed in the previous SAB report (SAB, 1995). However, there are a number of issues regarding the specifics of the calculations that the Panel believes need amplification. These include the incorporation of Monte

Carlo techniques and probability density functions to deal with uncertainties for each of the TEFs. There was also comment that this part of the report would benefit from additional editorial scrutiny.

Question 8 asked: "Have the available human data been adequately integrated with animal information in evaluating likely effect levels for the non-cancer endpoints discussed in the reassessment? Has EPA appropriately defined non-cancer adverse effects and the body burdens associated with them? Has EPA appropriately reviewed, characterized, and incorporated the recent epidemiologic evidence for the non-cancer risk assessment for human populations?" Dr. Weiss served as the lead discussant.

Most Members felt that the EPA deserved credit for pulling together a wide-ranging and diverse literature into a reasonable document. The Integrated Risk Summary presented conclusions drawn from the human and experimental literature in a reasonable manner. Basically, EPA used the human data as qualitative support for the observations of non-cancer endpoints in laboratory animals, but did not use them to calculate MOEs or any other quantitative measure of toxicity for dioxin. Given the uneven quality of the available human data and some seemingly conflicting findings, most Members of the Panel believe that this level of integration is, at present, appropriate to the task.

At this point, the Subcommittee resumed hearing comments from members of the public until all those registered to speak had done so. Dr. Lippman then adjourned the meeting at 6 pm, to resume the next day 8:30 am.

Thursday, November 2, 2000

Dr. Lippmann called the meeting to order at 8:30 am. He announced that the order of addressing the various questions would be altered in order to accommodate Dr. Greenlee who had to leave the meeting early because of a family emergency.

The Subcommittee then turned to Question 11, which asked: "Does the document clearly present the evolving approaches to estimating cancer risk (e.g., margin of exposure and the LED_{01} as a point of departure), as described in the EPA "Proposed Guidelines for Carcinogenic Risk Assessment" (EPA/600/P-92/003C; April 1996)? Is this approach equally as valid for dioxin-like compounds? Has EPA appropriately reviewed, characterized, and incorporated the recent epidemiological evidence for cancer risk assessment for human populations?" Dr. Graham served as lead discussant.

Most of the Subcommittee felt that the document provided a clear explanation of the evolving approaches to estimating cancer risk, and that the applicability to dioxin-like compounds depends upon the appropriateness of the TEQ approach in the specific value selected for implementation of that approach. The third element of this Charge question provoked considerable discussion. There were significant concerns about the reassessment document's interpretation and modeling of the recent epidemiological evidence. These concerns included the validity of the cancer slope factor, the document's treatment of the possible role of confounding in the three occupational cohorts studied, and

the issue of smoking as a confounder. Some Members were concerned about the validity of EPA's rationale for not doing dose response modeling on the Ranch Hand cohorts survey data, that the epidemiological data on dioxin-like compounds has not been reviewed rigorously in chapters 8 and 9, and that it would be difficult for the Subcommittee to comment on the classification of the dioxin-like compounds based upon the epidemiological evidence, since they had not reviewed the detailed epidemiological evidence. After considerable discussion of these issues, it became clear that no clear consensus would emerge, particularly on the issues relating to epidemiology.

The Subcommittee then addressed Question 9, which asked: "Do reviewers agree with the characterization of human developmental, reproductive, immunological, and endocrinological hazard? What, if any, additional assumptions and uncertainties should EPA embody in these characterizations to make them more explicit?" Dr. Kim served as lead discussant, in the absense of Dr. McConnell, who could not attend because of illness.

This was another area where there was a range of views among the Subcommittee membership. Most Members felt that the document, as written, was a logical presentation of the data on potential developmental, reproductive, immunological, and endocrinological hazards. However, the Subcommittee also noted that the question is broader in that it poses the question as to whether there is a human hazard for any of these endpoints. The summary statement in Section 6 of Part III regarding the human developmental, reproductive, immunological, and endocrinological hazards of dioxin appears to conclude that, although such hazards have not been conclusively demonstrated in humans, EPA presumes they can occur in humans because of their reported occurrence in laboratory animals and the presumed similarities in mechanisms between humans and laboratory animals. Although some Members believed that at least some of these endpoints have in fact been observed in human populations, other Members held that negative results in some high-exposure human cohorts is evidence against a human hazard for some endpoints, except for developmental toxicity. Most Members of the Panel agreed with the argument that occurrence in animals plus similarity of mechanism is a good argument for the assumption of hazard in humans. Some participants on the Panel believe that, because so little is known of the mechanisms of action in either animals or humans, it diminishes confidence in the extrapolation. At the same time, however, these Members recognize that such a situation is common in toxicology, and not confined merely to dioxin.

Question 12 asked: "Please comment on the presentation of the range of upper bound risks for the general population based on this reassessment. What alternative approaches should be explored to better characterize quantitative aspects of potential cancer risk? Is the range that is given sufficient, or should more weight be given to specific data sources?" Dr. Crump served as lead discussant.

The Panel agreed that, in general, the treatment of the range of upper bound risks obtained for the general population in this assessment is consistent with past EPA practice. The available data do not rule out a linear dose-response, and a supra-linear response seems implausible. Given this situation, the use of a linear response to define the upper bound is not inappropriate. The Subcommittee also agreed

that the human data are not adequate to define the dose response curve shape. The fact that the animal and human data predicted risks in the same range provides some support for the plausibility of the estimates. The Subcommittee discussed a number of suggestions regarding the calculation of the range and analyses that could more completely explore the range of upper bound risks. They noted that the only dose metric used to calculate ED_{01} from the epidemiology data was average lifetime body burden, whereas it would have been useful to see results using other dose metrics, particularly other metrics based on body burden. The Subcommittee also suggested that the analysis of the human data in Chapter 8 requires additional background exposition, and should organized better. The calculation of an ED_{01} from each of the three epidemiological studies are described in a single sentence that says only that a linear model was fit using Poisson regression. The membership felt that this was not an adequate description of the fitting process, and also noted that there was no description of how the results of the Poisson regression were converted to ED_{01} estimates. Some of the information in Chapter 10 presumably applies to the analyses in Chapter 8 as well, but this information needs to be incorporated in Chapter 8. Both upper and lower confidence limits on the ED_{01} would help to better characterize the range. Also, some Members thought that calculation of other ED, such as ED_{05}, would be useful. Some Panel Members expressed the view that Monte Carlo analyses would help to understand the range of potential risks, but others questions this suggestion, noting that, whereas such analyses can be helpful in expressing variability, they have less value in addressing fundamental uncertainty.

Question 10 asked: "Do you agree with the characterization in this document that dioxin and related compounds are carcinogenic hazards for humans? Does the weight-of-the-evidence support EPA's judgement concerning the listing of environmental dioxins as a likely human carcinogen?" Dr. Brown served as lead discussant.

Many Members addressed this issue in a lengthy discussion. Most of the Subcommittee agreed that causal associations have been established between exposures to some dioxin-like compounds and increased cancer incidence for some types of cancers in some species of laboratory animals. Those Members also agreed that the body of information was sufficient to satisfy the criterion for compelling evidence of carcinogenicity in laboratory animals, at least for TCDD. Most Subcommittee Members also agreed that the human epidemiologic study cited are not inconsistent with the finding of suggestive evidence for human carcinogenicity, but these Members differed in their confidence that the reported statistically significant associations can be concluded to be causal. Finally, most Members of the Subcommittee found persuasive EPA's arguments regarding the similarities in the mode of action between laboratory animals and humans. Therefore, the majority of the Subcommittee agreed that TCDD and probably some other dioxin-like compound satisfy EPA's criteria for human cancer hazard. Several Members were concerned, however, whether this characterization is indeed the message that EPA should be sending to the public. They noted that, although weight-of-evidence characterizations of the potential for human carcinogenicity should surely be seen as a continuum from a scientific perspective, the public probably responds qualitatively differently to human carcinogen than it does to the term suspected human carcinogen, probable human carcinogen, or presumptive human carcinogen. Moreover, the designation has significance in regulatory and litigation realms that do not recognize

strength- of-evidence as a continuum. Some Members did not think that TCDD, let alone some of the other dioxin-like compounds, deserve to be in the same category as cigarette smoking, asbestos, and radon. They cited a variety of deficiencies in the available human epidemiology, including questions about trends with exposure, inconsistency in elevated cancer rates, skepticism regarding the ability of an agent toaffect all cancers combined, and the potential for confounding by multiple non-dioxin risk factors.

Some Members were also concerned about inconsistencies between animal and human carcinogenicity observations. At least one Member felt, however, that the animal evidence was really overwhelming, revealing a multi-species, multi-strain, both sex carcinogen. This Member believes it had been characterized and was very strong, and that the human data, even on TCDD itself, could be considered to be at the least limited, and possibly even stronger than that. He pointed out that the IARC committee that reviewed the animal data, human data, and mechanistic data, concluded that it should be Group I. Another Member, however, noted that the reassessment document should make a much clearer and unequivocal statement about the Kociba study provided evidence of TCDD's anti-carcinogenicity with respect to mammary tumors, effects that cannot be explained away by weight loss, as suggested by EPA.

Question 13 asked: "Have the estimates of background exposures been clearly and reasonably characterized?" Dr. Thomas served as lead discussant.

The Subcommittee agreed that the overall estimates of background exposures have been clearly and reasonably characterized, but there were some important issues that called for comment. The Subcommittee was concerned about EPA's interpretation of the difference between the calculated tissue levels (based on dietary intake) versus the measured tissue levels as demonstrating declining dioxin levels in the environment. The Subcommittee felt that EPA had perhaps overstated the case supporting the assertion that tissue levels "appear to be declining." The Subcommittee also had concerns EPA's handling of the variability in the background exposures for the general population, and their assumption that the general population receives its food from a wide variety of sources throughout the country and therefore the impact of food from areas with particularly high dioxin levels will be diminished on the average.

Question 14 asked: "Has the relationship between estimating exposures from dietary intake and estimating exposure from body burden been clearly explained and adequately supported? Has EPA adequately considered available models for the low-dose exposure-response relationships (linear, threshold, "J" shaped)?" Dr. McKone was the lead discussant.

The Subcommittee addressed this question in two parts. The first component dealt with whether the relationship between estimating exposures from dietary intake and estimating exposure from body burden has been clearly explained and adequately supported. The Subcommittee agreed that this relationship is clearly explained and adequately supported. They noted, however, that the uncertainty in

the parameters and the model inputs should be more clearly emphasized. Due to these uncertainties, the difference between the measured and calculated tissue levels should not be assumed to be significant. The Members reached general agreement that the Agency has used a reasonable approach to estimate daily uptake of dioxin and dioxin-like compounds.

The second component addressed dealt with low-dose exposure responses. The Subcommittee decided that the actual shape of the low-dose exposure response relation cannot yet be determined from the available data. They agreed that the EPA complied with the 1995 SAB review's request that EPA evaluate evidence related to low dose exposures. Finally, some Members suggested the discussion in the relevant sections of the reassessment document should be more complete and consider what is known about the promoter-like characteristics of 2,3,7,8 TCDD.

Question 15 asked: "Have important 'special populations' and age-specific exposures been identified and appropriately characterized?" Dr. Kim served as lead discussant.

The Subcommittee felt that EPA appropriately identified several populations as having the potential to be highly exposed. These populations include nursing infants, individuals with unique diets, occupationally exposed individuals, cigarette smokers, and individuals who may live near significant sources of dioxin. Some Subcommittee Members noted that biologically susceptible populations could include individuals that are at increased risk because of age or gender, or some other population characteristic-specific effect, as well as those individuals that could be genetically susceptible. The Members agreed that the Reassessment Document did a good job of identifying those at increased risk because of demographic characteristics, but there was very limited information available on genetic susceptibility; they also suggested that some further discussion of genetic predisposition and special dietary preferences or limitations would be desirable.

Question 16 asked: "Is the characterization of increased or decreased childhood sensitivity to possible cancer and non-cancer outcomes scientifically supported and reasonable? Is the weight of the evidence approach appropriate?" Dr. Lambert served as lead discussant.

The Subcommittee agreed that the draft Reassessment document's characterization of childhood sensitivity to possible cancer and non-cancer outcomes should be improved . They noted that, in regard to cancer endpoints, the Agency accurately portrays the lack of studies that can address this question. However, noting the SAB's review of the proposed cancer guidelines for children, the Members suggested that, when a chemical's mechanism of action is proposed and discussed, the Agency should identify all the critical steps in the mechanism and identify what is known about these steps (proteins, receptors) in the developing human.

Question 17 asked: "Has EPA adequately characterized how nursing affects short-term and long-term body burdens of dioxins and related compounds?" Dr. Kim served as lead.

The Subcommittee was pleased that EPA addressed this issue, which was not included in the 1994 document. The Members recommended that the exposure scenarios be extended to address those who engage in more prolonged breast feeding. The Subcommittee also noted that EPA should incorporate information about blood levels from the German studies into the first paragraph of the relevant section of the Risk Summary in order to place these data into context. The Subcommittee found the characterization of cancer health risks to nursing infants to be adequate, but some Members feel that EPA could have been more direct in noting that a putative human carcinogen or tumor promoter such as dioxin will not result in higher lifetime risks of cancer for exposure in childhood as compared with exposures during adulthood, even after adjusting for the temporarily higher doses received during childhood. Lastly, the Subcommittee was somewhat surprised that EPA provided only a minimal characterization of non-cancer health risks for infants and children (especially considering the effort devoted to cancer).

Question 18 asked: "Does the summary and analysis support the conclusion that enzyme induction, changes in hormone levels, and indicators of altered cellular function seen in humans and laboratory animals, represent effects of unknown clinical significance, but they may be early indicators of toxic response?" In the absence of Dr. Greenlee, Dr. Albert served as the lead discussant.

The Subcommittee generally supported the position that non-stochastic processes like those induced by dioxin are graded in character. At higher doses, there are strong multiple effects. With diminishing dose levels, the range of effects narrows and their intensity decreases. Some Members noted that small effects, like perturbations in enzyme and hormone levels, may be anticipated at low doses, and there may be ambiguity as to whether these effects are adaptive or compensator; in either case they may not necessarily be detrimental. Other Members felt that, in the absence of information to the contrary, that they should be regarded as evidence of mild toxicity. Overall, the Subcommittee was divided about the health significance of such changes. Several Members were uncomfortable with the position that effects such as enzyme induction, changes in hormone levels and indicators of altered cellular function may be early indicators of toxic response.

Question 19 asked "Has the short summary statement in the risk and hazard characterization on page other that 107 adequately captured the important conclusions and the areas where further evaluation is needed. What additional points should be made in this short statement?" Dr. Albert served as lead discussant.

The Subcommittee commented that the Summary Statement is a very important part of the document, since it is the only place that non-technical readers, including risk managers, can get an overview of the assessment and its conclusions. Some Members noted that the Summary Statement was too one-sided in failing to adequately present the full range of legitimate opinion about the interpretation of the evidence for dioxin as a human carcinogen. They feel that the basic conclusion of the risk assessment is a flat-out assertion that dioxin is a human carcinogen and that current body burdens are dangerous, and that this assertion should be tempered with further information vis-avis uncertainty, etc. The

Subcommittee also recommends that complete reliance on the upper confidence limit (based on EPA's standard models and defaults) for quantitative risk assessment of cancer risks also needs to be tempered. The Summary might also point out that with a receptor mediated cancer process, the best estimate of risk from the linear non-threshold model is already an "upper limit."

Question 20 asked: "Are these sources adequately described and are the relationships to exposure adequately explained?" Dr. Thomas served as lead discussant.

The Subcommittee had high praise for the the Inventory of Dioxin Sources, and commended the Agency for the effort. This praise notwithstanding, the Subcommittee identified some problems with the presentation of the inventory results, calling them somewhat confusing. The Members identified two major problems: a) the exclusion of the so-called "unquantified" sources from the main description of the sources; and b) the lack of consistency of the Summary Document (Part III) with the Sources Inventory.

Following final discussion of report preparation, the Chair thanked the various participants on the Subcommittee, and the EPA staff present, and adjourned the meeting at 4:05pm.

I certify that these minutes are accurate to the best of my knowledge.

/s/

Dr. Morton Lippmann
Chair, Dioxin Reassessment Review Subcommittee

/s/

Mr. Samuel Rondberg
Designated Federal Officer

ATTACHMENTS

ATTACHMENT G

Public Speakers:
(in order of appearance)

Gary Kayajanian
Alan Lockwood
Devra Davis
Leon Bradlow
Dimitrios Trichopoulos
Dr. Joe Thornton
Dr. Clifford Firstenberg
Dr. Thomas Sutter
Dr. Barbara Peterson
Lesa Aylward
Marcie Francis
Ellen Silbergeld
Thomas B. Starr
Michael Gough
Steven Milloy
James Brown
Kenneth Fish
Russell Keenan
Tim King
Laura Valeriano
Kimberly Kelly
Arnold Schecter
Tom Webster
Stephen Lester
Donald Millar
Vernell Cutter
Charlotte Brody
Susan Chang (in place of Mary Richter)
Sam McClure
Brianey Schwan
Linda Schwartz
Ms. Scott (not present)
Tracey Easthope
Linda Noble
Bill Smedley

Charlotte Caldwell
Esther Nahgahnub
Kenneth Bradshaw
Pamela Miller (spoke twice)
Dennis Lee
Julie Filapek
Bill Walsh
Pamela Miller (2nd time)
Rick Weidman (in place of Dr. Schwartz)
Tamara Maschino
Don Tillett
David Wallinga